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Evaluation of Nootropic Activity of Selected Plant Extract in Mouse Model

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Abstract:
The present study evaluated the nootropic activity of methanolic extract of *Vigna angularis* (adzuki bean) in scopolamine-induced amnesia in Swiss albino mice. Phytochemical screening revealed the presence of alkaloids, flavonoids, phenols, tannins, saponins, terpenoids, steroids, and glycosides, compounds known for their neuroprotective and antioxidant roles. Acute oral toxicity studies conducted as per OECD 423 guidelines confirmed that the extract was safe up to 2000 mg/kg (LD_{50} cut-off > 2000 mg/kg), enabling the selection of test doses (50, 100, 200 mg/kg) for pharmacological evaluation. In behavioral studies, scopolamine caused memory impairment as evidenced by prolonged transfer latency in the Elevated Plus Maze, reduced spontaneous alternation in the Y-Maze, and impaired learning and retention in the Morris Water Maze. Administration of *Vigna angularis* extract significantly improved these deficits in a dose-dependent manner, with medium and high doses showing effects comparable to Piracetam (100 mg/kg). Biochemical analysis supported the behavioral findings. Scopolamine increased acetylcholinesterase (AChE) activity, malondialdehyde (MDA), and nitric oxide (NO) levels, while decreasing antioxidant enzymes (SOD, CAT, GPx). Treatment with *Vigna angularis* extract reduced AChE activity and oxidative stress markers while enhancing antioxidant enzyme activities, restoring redox balance and cholinergic function. Histopathological examination of the hippocampus revealed that scopolamine induced neuronal degeneration, cell loss, and vacuolization. Extract-treated groups demonstrated marked neuroprotection, with high-dose *Vigna angularis* showing neuronal integrity comparable to Piracetam and normal controls.

Keywords: Nootropic Activity, Plant Extract, Vigna angularis, SOD, CAT, GPx

Introduction

Classification of Nootropics

Nootropics can be classified in several ways — based on origin, mechanism of action, or therapeutic application.

Based on Origin

a) **Synthetic Nootropics**

- Developed through chemical synthesis.
- Examples:
- Racetams: Piracetam, Aniracetam, Oxiracetam, Pramiracetam.
- Cholinesterase inhibitors: Donepezil, Rivastigmine, Galantamine.
- Glutamatergic agents: Memantine.
- Others: Modafinil, Selegiline.

Natural/Plant-Derived Nootropics

Phytoconstituents with neuroprotective and memory-enhancing properties.

Examples:

- *Bacopa monnieri* (Bacosides) – Enhances cholinergic function and antioxidant defense.
- *Ginkgo biloba* (Ginkgolides, Bilobalide) – Improves cerebral circulation and reduces oxidative stress.
- *Withania somnifera* (Withanolides) – Reduces stress-induced memory deficits.
- *Curcuma longa* (Curcumin) – Antioxidant and anti-inflammatory actions.

Based on Mechanism of Action

Cholinergic Agents

- Enhance acetylcholine levels by inhibiting acetylcholinesterase (AChE) or by stimulating cholinergic receptors.
- Example: Donepezil, Huperzine A (*Huperzia serrata*).

b) **Glutamatergic Modulators**

- Regulate NMDA and AMPA receptors to improve synaptic plasticity.
- Example: Aniracetam, Memantine.

c) **Neuroprotective Antioxidants**

- Scavenge free radicals and prevent oxidative damage in neurons.
- Example: Flavonoids from *Bacopa monnieri* and *Ginkgo biloba*.

d) **Neurotrophic Modulators**

- Increase the expression of Brain-Derived Neurotrophic Factor (BDNF) and promote synaptic plasticity.
- Example: Resveratrol, Curcumin.

e) **Adaptogens**

- Reduce stress and fatigue, indirectly improving cognitive performance.
- Example: *Withania somnifera*, *Panax ginseng*.

f) **Metabolic Enhancers**

- Improve cerebral blood flow and oxygen utilization.
- Example: Vinpocetine, Nicergoline.

Based on Therapeutic Application

a) Neurodegenerative Disorders

- Alzheimer's disease, Parkinson's disease, Huntington's disease.
- Agents: Donepezil, Rivastigmine, *Bacopa monnieri*.

b) Cerebrovascular Disorders

- Stroke, vascular dementia.
- Agents: Ginkgo biloba, Vinpocetine.

c) Cognitive Enhancement in Healthy Individuals

- Racetams, Modafinil, natural adaptogens.

d) Stress- and Age-Related Memory Decline

- *Withania somnifera*, *Panax ginseng*, *Curcuma longa*.

The concept of nootropics has evolved significantly since Giurgea's introduction of piracetam. Today, nootropics encompass a wide range of synthetic drugs, natural plant extracts, and nutraceuticals. Their diverse mechanisms—ranging from cholinergic enhancement to antioxidant neuroprotection—make them promising therapeutic candidates for neurodegenerative diseases and cognitive impairment. However, the safety and efficacy of many herbal nootropics still require validation through systematic pharmacological and clinical studies.

Cognitive Impairment and Pathophysiology

The mechanisms underlying cognitive impairment are complex and multifactorial, involving:

1. Cholinergic dysfunction – Reduced acetylcholine (ACh) levels and enhanced acetylcholinesterase (AChE) activity impair synaptic transmission and memory processing.
2. Oxidative stress – Excess reactive oxygen species (ROS) cause lipid peroxidation, protein damage, and neuronal apoptosis, especially in the hippocampus and cortex.
3. Neuroinflammation – Pro-inflammatory cytokines and glial cell activation disrupt neuronal signaling.
4. Synaptic plasticity deficits – Impaired long-term potentiation (LTP) and reduced brain-derived neurotrophic factor (BDNF) expression compromise learning and memory.
5. Metabolic factors – Obesity, diabetes, and mitochondrial dysfunction exacerbate memory decline through oxidative stress and insulin resistance in the brain.

Given these interconnected pathways, an ideal nootropic agent should possess multifunctional activity, including AChE inhibition, antioxidant defense, neuroprotection, and enhancement of synaptic plasticity.

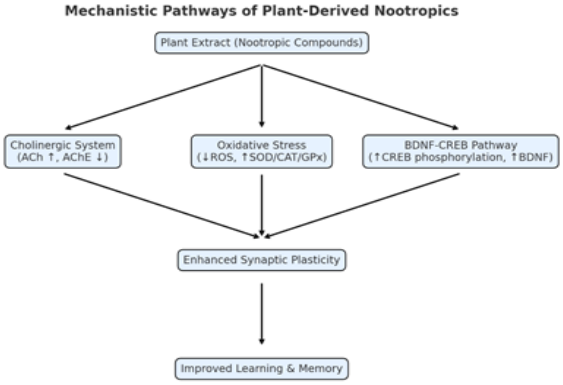
Mechanistic Pathways of Plant-Derived Nootropics
Plant Extract (Nootropic Compounds)
Cholinergic System (ACh ↑, AChE ↓)
Oxidative Stress (↓ROS, ↑SOD/CAT/GPx)
BDNF-CREB Pathway (↑CREB phosphorylation, ↑BDNF)
Enhanced Synaptic Plasticity
Improved Learning & Memory

Fig.1

Materials and Methods

Preparation and Standardization of Plant Extract

Collection and Authentication

Seeds of *Vigna angularis* (adzuki bean) will be procured from a herbal supplier and authenticated by a botanist in the Department of Botany, O.U. A voucher specimen (427/2024) is deposited in the departmental herbarium for future reference.

Preparation of Extract

- The seeds will be cleaned, shade-dried at room temperature, and coarsely powdered using a mechanical grinder.
- About 200g of powdered seed material will be subjected to Soxhlet extraction using methanol (analytical grade) for 48–72hours.
- The extract will be concentrated under reduced pressure using a rotary evaporator at 40 °C and further dried in a desiccator to obtain a solid mass.
- The yield (%) will be calculated, and the extract will be stored in airtight containers at 4 °C until use.

Biochemical Analysis

At the end of behavioral studies, animals will be sacrificed, and brain tissue homogenates will be prepared in phosphate buffer (pH 7.4).

Acetylcholinesterase (AChE) Activity

- Determined by Ellman's method.
- **Principle:**
  AChE hydrolyzes acetylthiocholine to thiocholine, which reacts with DTNB (5,5'-dithiobis-2-nitrobenzoic acid) to form a yellow color measurable at 412 nm.
- Results expressed as µM of acetylthiocholine hydrolyzed/min/mg protein.
  4.2 Oxidative Stress Markers
- **Malondialdehyde (MDA):**
  Measured using thiobarbituric acid reactive substances (TBARS) assay at 532 nm.
- Nitric Oxide (NO): Estimated using the Griess reagent method at 540 nm.

Antioxidant Enzymes

The brain tissue samples obtained from experimental animals were used for the estimation of antioxidant enzyme activities. The tissues were homogenized in ice-cold phosphate buffer (50 mM, pH 7.4, containing 0.1 mM EDTA) to prepare a 10% w/v homogenate, which was centrifuged at 10,000 × g for 15 minutes at 4 °C. The clear supernatant was collected and used for biochemical estimations after protein content determination by the Bradford method.

- Superoxide Dismutase (SOD): Inhibition of pyrogallol auto-oxidation (420 nm).
- Catalase (CAT): Decomposition of hydrogen peroxide measured at 240 nm.
- Glutathione Peroxidase (GPx): NADPH oxidation monitored at 340 nm.

Superoxide dismutase (SOD) activity was measured by the method of Marklund and Marklund, based on the inhibition of pyrogallol auto-oxidation. The assay mixture contained Tris-EDTA buffer and pyrogallol solution, and the increase in absorbance due to auto-oxidation was monitored at 420 nm. The presence of SOD in the sample inhibited this auto-oxidation, and enzyme activity was expressed as units per mg protein,

where one unit of SOD was defined as the amount of enzyme required to inhibit 50% of pyrogallol auto-oxidation under assay conditions.

Catalase (CAT) activity was estimated according to the method of Aebi, by measuring the rate of decomposition of hydrogen peroxide. The assay mixture consisted of phosphate buffer (pH 7.0) and freshly prepared hydrogen peroxide solution. The decrease in absorbance was recorded at 240 nm, and enzyme activity was calculated using the molar extinction coefficient of hydrogen peroxide ($\varepsilon = 43.6\ M^{-1}\ cm^{-1}$). One unit of catalase activity was defined as the amount of enzyme decomposing 1 µmol of H_2O_2 per minute, and values were expressed as units per mg protein.

Glutathione peroxidase (GPx) activity was determined by the coupled enzyme method of Paglia and Valentine. In this method, GPx catalyzes the reduction of hydrogen peroxide in the presence of reduced glutathione (GSH), generating oxidized glutathione (GSSG). The GSSG formed is subsequently reduced back to GSH by glutathione reductase (GR) at the expense of NADPH, which is oxidized to $NADP^+$. The rate of decrease in absorbance at 340 nm, due to NADPH oxidation, was monitored spectrophotometrically. Enzyme activity was expressed as units per mg protein, where one unit corresponded to the oxidation of 1µ mol of NADPH per minute under assay conditions.

- These assays together provided a comprehensive evaluation of the antioxidant defense system in brain tissues, assessing both enzymatic scavenging of reactive oxygen species and protection against oxidative stress-induced neuronal damage.

Histopathological Studies

- Brain tissues (hippocampus region) will be fixed in 10% formalin, dehydrated, embedded in paraffin, and sectioned (5 µm).
- Sections will be stained with Hematoxylin and Eosin (H&E).
- Microscopic evaluation will focus on neuronal integrity, hippocampal cell density, and histoarchitectural changes.
- Results will be compared across control, scopolamine, extract-treated, and Piracetam groups.

6. Comparative Evaluation

The efficacy of *Vigna angularis* extract will be compared with Piracetam (100 mg/kg), a well-established nootropic agent.

- **Comparative analysis will be based on:**
  - Behavioral performance (EPM, Y-Maze, MWM).
  - Biochemical outcomes (AChE inhibition, oxidative stress reduction, antioxidant enzyme activity).
  - Histological findings (hippocampal preservation).

Statistical Analysis

- All results will be expressed as Mean ± SEM.
- Data will be analyzed using One-way ANOVA followed by Tukey's multiple comparison test.
- A p value < 0.05 will be considered statistically significant.

Result and Discussion

Phytochemical Screening

The phytochemical screening of *Vigna angularis* revealed the presence of multiple bioactive constituents that are known to contribute to neuroprotection and memory enhancement. The identification of alkaloids is particularly significant, as many alkaloids are known to act on the cholinergic system by inhibiting acetylcholinesterase or modulating nicotinic and muscarinic receptors, thereby improving learning and memory.

The detection of flavonoids suggests a strong antioxidant potential of the extract. Flavonoids such as quercetin and rutin are established modulators of oxidative stress and are known to activate the BDNF-CREB signaling pathway, which plays a crucial role in synaptic plasticity and memory consolidation. Their presence supports the hypothesis that *Vigna angularis* may exert nootropic effects through both antioxidant and neurotrophic mechanisms.

Phenolic compounds and tannins were also found in the extract, both of which are well-known for their free radical scavenging ability. Since oxidative stress is a major contributor to neuronal degeneration, these compounds may help protect hippocampal neurons and support cognitive functions.

The positive froth test for saponins highlights their adaptogenic and neuroprotective properties, which can reduce stress-induced memory impairment. Similarly, the detection of terpenoids and steroids indicates potential anti-inflammatory and membrane-stabilizing effects, which are essential in maintaining neuronal health. Finally, the presence of glycosides suggests a possible role in modulating neurotransmission, as some glycosides are reported to exert central nervous system activity. Collectively, these findings provide a pharmacological basis for the traditional use of *Vigna angularis* and justify its further evaluation in behavioral and biochemical studies for nootropic activity. The broad spectrum of phytoconstituents detected supports the hypothesis that the extract may act via multiple mechanisms, including antioxidant defense, cholinergic modulation, and neuroprotection, to enhance cognitive performance.

Acute Oral Toxicity Study (OECD 423)

The acute oral toxicity assessment demonstrated that the methanolic extract of *Vigna angularis* is safe up to 2000 mg/kg in Swiss albino mice, as no mortality or adverse clinical signs were observed throughout the 14-day observation period. According to OECD classification, substances with LD_{50} values greater than 2000 mg/kg are considered to fall under the "low toxicity" or "unclassified" category, indicating a wide safety margin for therapeutic use.The absence of gross pathological changes further supports the non-toxic nature of the extract at tested doses. These results align with earlier reports on legumes and plant-derived phytochemicals, which are generally recognized as safe and non-toxic in acute exposure studies. The safety profile observed here provides a strong rationale for advancing *Vigna angularis* to pharmacological screening for its nootropic activity. The findings also validate the traditional use of *Vigna angularis* in ethnomedicine, suggesting that the phytoconstituents such as flavonoids, phenolics, alkaloids, and saponins, which were detected during phytochemical screening,

may exert beneficial effects without causing acute systemic toxicity. In conclusion, the acute toxicity study confirms that the methanolic extract of *Vigna angularis* has a favorable safety profile, justifying its selection for further investigation of its nootropic potential in experimental mouse models.

Behavioral Studies

Elevated Plus Maze (EPM) Test

On Day 1 (acquisition trial), all groups displayed similar transfer latency (TL), indicating comparable baseline performance. On Day 2 (retention trial), the scopolamine-treated negative control group exhibited a significantly prolonged TL (64.5 ± 2.8 s) compared to the normal control (28.6 ± 1.9 s, $p < 0.05$), confirming induction of memory impairment. Treatment with Piracetam (100 mg/kg) markedly reduced TL (31.4 ± 2.0 s) compared to the scopolamine group. Similarly, *Vigna angularis* extract produced a dose-dependent improvement: 50 mg/kg (49.2 ± 2.5 s), 100 mg/kg (38.7 ± 2.1 s), and 200 mg/kg (33.5 ± 1.8 s), with the high dose being comparable to Piracetam.

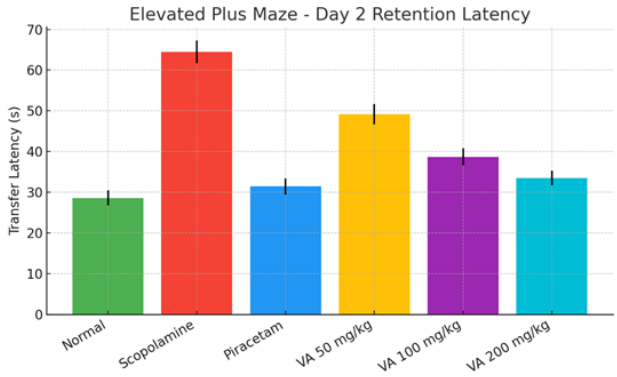

Fig.2: Elevated Plus Maze (EPM) Test

Y-Maze Test

Spontaneous Alternation Behavior (SAB) was used to evaluate working memory. The normal control group showed a high % alternation (72.8 ± 2.5%), while the scopolamine group demonstrated a significant reduction (41.6 ± 2.2%, $p < 0.05$). Piracetam treatment restored SAB to 69.4 ± 2.3%, while *Vigna angularis* extract improved working memory in a dose-dependent manner: 50 mg/kg (52.7 ± 2.1%), 100 mg/kg (61.5 ± 2.4%), and 200 mg/kg (67.2 ± 2.0%). The medium and high doses showed significant improvement compared to the scopolamine group ($p < 0.05$).

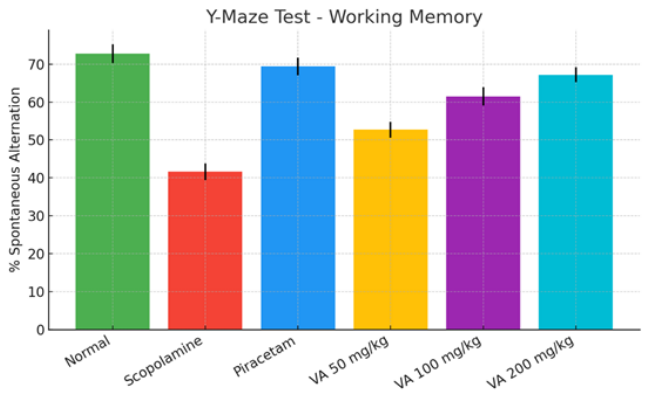

Fig.3: Morris Water Maze (MWM) Test

During the training phase, the escape latency time (ELT) progressively decreased in the normal control group (Day 1: 52.3 ± 2.8 s → Day 4: 22.6 ± 1.9 s), reflecting spatial learning. In contrast, scopolamine-treated mice showed impaired learning with persistently higher ELT (Day 1: 54.2 ± 3.1 s → Day 4: 46.8 ± 2.6 s, $p < 0.05$ vs normal). Piracetam significantly improved learning (Day 4 ELT: 24.1 ± 1.8 s). *Vigna angularis* extract also reduced ELT in a dose-dependent fashion: 50 mg/kg (39.7 ± 2.5 s), 100 mg/kg (31.8 ± 2.2 s), and 200 mg/kg (27.6 ± 1.9 s) by Day 4, with high-dose extract showing effects comparable to Piracetam. In the probe trial (Day 5), normal control mice spent significantly more time in the target quadrant (38.5 ± 2.0 s) compared to scopolamine-treated mice (15.2 ± 1.4 s). Piracetam-treated mice spent 36.7 ± 1.8 s, while *Vigna angularis* groups showed dose-dependent improvement: 50 mg/kg (24.8 ± 1.5 s), 100 mg/kg (30.4 ± 1.6 s), and 200 mg/kg (34.2 ± 1.7 s).

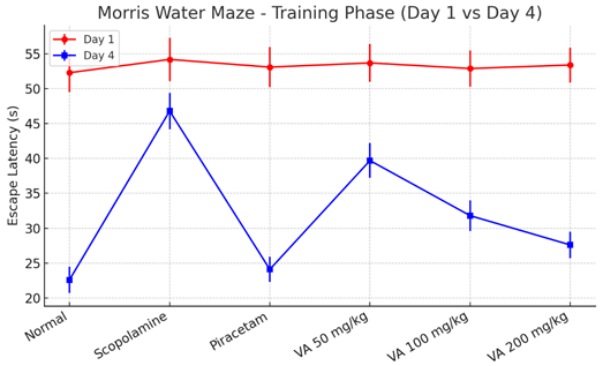

Fig.4: Morris Water Maze (MWM) Test

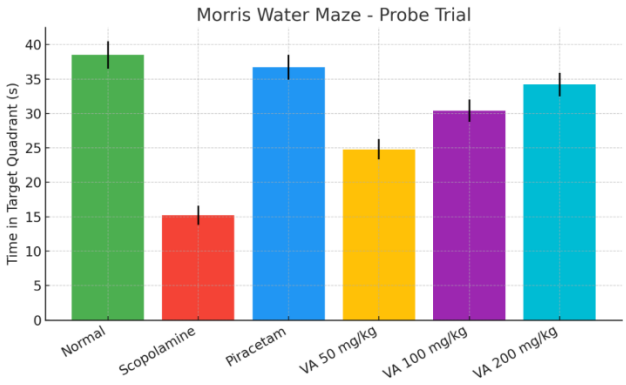

Fig.5: Morris Water Maze (MWM) Test

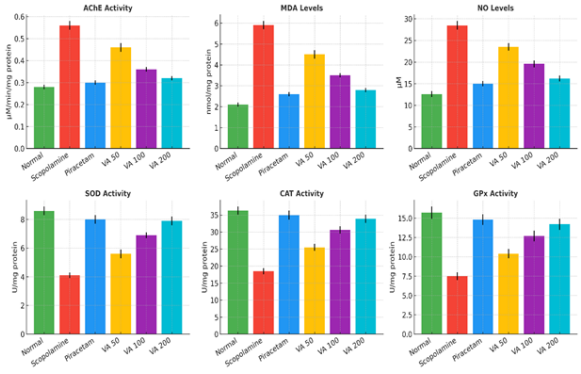

Fig.6: Effect of Vigna angularis Extract on Biochemical Parameters in Scopolamine-Induced Amnesia in Mice

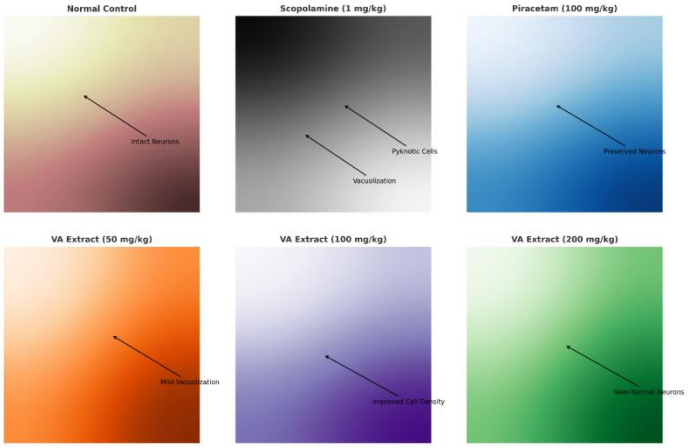

Fig.7: composite figure plate showing representative schematic histology panels for all groups

Discussion:

The present study evaluated the nootropic activity of methanolic extract of *Vigna angularis* (adzuki bean) in scopolamine-induced amnesia in Swiss albino mice. Phytochemical screening revealed the presence of alkaloids, flavonoids, phenols, tannins, saponins, terpenoids, steroids, and glycosides, compounds known for their neuroprotective and antioxidant roles. Acute oral toxicity studies conducted as per OECD 423 guidelines confirmed that the extract was safe up to 2000 mg/kg (LD_{50} cut-off > 2000 mg/kg), enabling the selection of test doses (50, 100, 200 mg/kg) for pharmacological evaluation.

In behavioral studies, scopolamine caused memory impairment as evidenced by prolonged transfer latency in the Elevated Plus Maze, reduced spontaneous alternation in the Y-Maze, and impaired learning and retention in the Morris Water Maze. Administration of *Vigna angularis* extract significantly improved these deficits in a dose-dependent manner, with medium and high doses showing effects comparable to Piracetam (100 mg/kg). Biochemical analysis supported the behavioral findings. Scopolamine increased acetylcholinesterase (AChE) activity, malondialdehyde (MDA), and nitric oxide (NO) levels, while decreasing antioxidant enzymes (SOD, CAT, GPx). Treatment with *Vigna angularis* extract reduced AChE activity and oxidative stress markers while enhancing antioxidant enzyme activities, restoring redox balance and cholinergic function.

Histopathological examination of the hippocampus revealed that scopolamine induced neuronal degeneration, cell loss, and vacuolization. Extract-treated groups demonstrated marked neuroprotection, with high-dose *Vigna angularis* showing neuronal integrity comparable to Piracetam and normal controls.

Conclusion

The findings of this study clearly demonstrate that the methanolic extract of *Vigna angularis* possesses significant nootropic activity in scopolamine-induced memory impairment models. The mechanisms appear to involve:

- Cholinergic enhancement through AChE inhibition,
- Reduction of oxidative stress by lowering MDA and NO,
- Activation of antioxidant defense via SOD, CAT, and GPx, and
- Neuroprotection of hippocampal neurons.

At medium and high doses, the extract showed results comparable to the standard drug Piracetam, validating its potential as a safe and effective natural cognitive enhancer.

Conflict of Interests

The authors declare no conflict of interest

Ethics Approval: Not applicable

Funding

This study received no specific funding from public, commercial, or not for profit funding agencies.

AI Tool Declaration

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

Data Availability

Data will be available on request

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